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Mutationally Activated PIK3CA<sup>H1047R</sup> Cooperates with BRAF<sup>V600E</sup> to Promote Lung Cancer Progression
Christy L. Trejo, Shon Green, Victoria Marsh, Eric A. Collisson, Gioia Iezza, Wayne A. Phillips, and Martin McMahon

Précis: These findings deepen the in vivo evidence that MAPK and PI3K signaling cooperates in mediating the development and progression of KRAS-mutated lung cancer, suggesting combination therapies to treat this disease.

Antitumor Efficacy of a Monoclonal Antibody That Inhibits the Activity of Cancer-Associated Carbonic Anhydrase XII
Gabor Gondi, Josef Mysliwietz, Alzbeta Hulikova, Jian Ping Jen, Pawel Swietach, Elisabeth Kremmer, and Reinhard Zeidler

Précis: This study offers a preclinical proof-of-concept for immune targeting a cell surface carbonic anhydrase that is widely expressed in human cancer as a general therapeutic strategy.

Photodynamic Therapy of Murine Mastocytoma Induces Specific Immune Responses against the Cancer/Testis Antigen P1A
Pawel Mroz, Fatma Vatansever, Angelika Muchowicz, and Michael R. Hamblin

Précis: Effective photodynamic therapy used to treat certain cancers may act as antigen-specific immunotherapy.

Bispecific Antibody to ErbB2 Overcomes Trastuzumab Resistance through Comprehensive Blockade of ErbB2 Heterodimerization
Bohua Li, Yanchun Meng, Lei Zheng, Xumin Zhang, Qing Tong, Wenlong Tan, Shi Hu, Hui Li, Yang Chen, Jinjing Song, Ge Zhang, Lei Zhao, Dapeng Zhang, Sheng Hou, Weizhu Qian, and Yajun Guo

Précis: Using a bispecific antibody to block ErbB2/HER2 heterodimerization on the surface of breast cancer cells may provide a strategy to overcome resistance to Herceptin that remains a major clinical challenge in breast cancer patients.

A Small-Molecule Blocking Ribonucleotide Reductase Holoenzyme Formation Inhibits Cancer Cell Growth and Overcomes Drug Resistance

Précis: These findings address deficiencies in existing drugs that block ribonucleotide reductase, offering preclinical validation of a promising new class of inhibitors against this valid target that could find broad use to treat many human cancers.

MYC Phosphorylation at Novel Regulatory Regions Suppresses Transforming Activity
Amanda R. Waylishen, Michelle Chan-Seng-Yue, Christina Rros, Dharmandra Dingar, William B. Tu, Manpreet Kalkat, Pak-Kei Chan, Peter J. Mullen, Ling Huang, Natalie Meyer, Brian Raught, Paul C. Boutros, and Linda Z. Penn

Précis: MYC phosphorylation mutants with super-transforming activity that were identified in this study point the way toward new therapeutic targets to attack MYC by a backdoor approach.

TIG1 Promotes the Development and Progression of Inflammatory Breast Cancer through Activation of Axl Kinase
Xiaoping Wang, Hitomi Saso, Takayuki Iwamoto, Weiya Xia, Yun Gong, Lajos Pusztai, Wendy A. Woodward, James M. Reuben, Steven L. Warner, David J. Bears, Gabriel N. Hortobagyi, Mien-Chie Hung, and Naoto T. Ueno

Précis: These findings provide key new insights into the molecular pathobiology of the most aggressive form of breast cancer, rationalizing the Axl receptor signaling pathway as a therapeutic target for treatment of this lethal disease.

Nitric Oxide Production Upregulates Wnt/β-Catenin Signaling by Inhibiting Dickkopf-1
Qiang Du, Xinglu Zhang, Quan Liu, Xianghong Zhang, Christian E. Bartels, and David A. Geller

Précis: In addressing the complex role of nitric oxide in cancer, this study furthers evidence of an oncogenic contribution that is mediated by a mechanism that stimulates Wnt/β-catenin signaling, a central pathway for carcinogenesis.

Correction: Breast Tumor Kinase (Brk/PTK6) Is a Mediator of Hypoxia-Associated Breast Cancer Progression

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ABOUT THE COVER

miR-153 leads to increased invasiveness in colorectal cancer. Using mouse tumor xenografts, it was found that colorectal tumors with inhibition of miR-153 show a clean edge of tumor spheroid and fewer invasive fronts into the surrounding stroma (magnification, ×400) in contrast to controls with a more locally invasive tumor phenotype. For details, see article by Zhang and colleagues on page 6435.
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